

**Pending Claims**

This listing of claims will replace all prior versions, and listings, of claims in the application:

Claims 1-28 (canceled).

- Claim 29. (new) A method for treating a mammal to resist early graft failure comprising,
- a) introducing into cells of a graft from the mammal an effective amount of at least one nucleic acid encoding at least one of the following agents: endothelial cell protein C receptor (EPCR), thrombomodulin (TM), NF- $\kappa$ B inhibitor, or a functional fragment thereof; provided that when the agent is thrombomodulin, the nucleic acid further encodes at least one of the endothelial cell protein C receptor (EPCR) or the NF- $\kappa$ B inhibitor, wherein the introducing is performed *ex vivo* or by direct injection into the graft,
  - b) expressing the agent in the cells to increase activated protein C (APC); and
  - c) increasing the APC in the graft cells sufficient to resist the early graft failure,
- wherein,
- (i) the functional fragment of the EPCR has at least about 85% of the protein C activation activity of human EPCR,
  - (ii) the functional fragment of the TM has at least about 85% of the thrombin binding activity of human thrombomodulin, and
  - (iii) the functional fragment of the NF- $\kappa$ B has at least about 90% of the activity of I $\kappa$ B.

- Claim 30. (new) A method for engineering a vascular graft of a mammal to resist early failure, the method comprising:

- a) introducing into cells of the graft from the mammal an effective amount of at least one nucleic acid encoding at least one of the following agents: endothelial cell protein C receptor (EPCR), thrombomodulin (TM), NF- $\kappa$ B inhibitor, or a functional fragment thereof; provided that when the agent is thrombomodulin, the nucleic acid further encodes at least one of the endothelial cell protein C receptor

(EPCR) or the NF- $\kappa$ B inhibitor, wherein the introducing is performed *ex vivo* or by direct injection into the graft,  
b) expressing the agent in the cells to increase activated protein C (APC); and  
c) increasing the APC in the graft sufficient to produce the engineered vascular graft,

wherein,

- (i) the functional fragment of the EPCR has at least about 85% of the protein C activation activity of human EPCR,
- (ii) the functional fragment of the TM has at least about 85% of the thrombin binding activity of human thrombomodulin, and
- (iii) the functional fragment of the NF- $\kappa$ B has at least about 90% of the activity of I $\kappa$ B.

Claim 31. (new) The method of claim 29, wherein the method further comprises transplanting the graft into the mammal.

Claim 32. (new) The method of claim 29, wherein prior to step a) of the method, the graft is transplanted into the mammal.

Claim 33. (new) The method of claim 29, wherein the method is performed on the graft *in vivo*.

Claim 34. (new) The method of claim 31, wherein the transplanted vascular graft has sufficient APC activation as determined by a standard protein C assay to prevent or treat early graft failure.

Claim 35. (new) The method of claim 34, wherein the level of protein C activation as determined by a standard protein C detection assay of the treated graft is at least about one order of magnitude higher than a control vessel.

Claim 36. (new) The method of claim 35, wherein the higher protein C level of the treated vascular graft is detectable for at least about a week.

Claim 37. (new) The method of claim 34, wherein the early graft failure is accompanied by thrombosis.

Claim 38. (new) The method of claim 29, wherein the nucleic acid is inserted into a cassette.

Claim 39. (new) The method of claim 38, wherein the cassette includes a promoter.

Claim 40. (new) The method of claim 39, wherein the cassette is inserted into a vector.

Claim 41. (new) The method of claim 40, wherein the vector comprises sequence from an adenovirus, retrovirus, or adeno-associated virus.

Claim 42. (new) The method of claim 41, wherein the vector is a replication defective adenovirus.

Claim 43. (new) The method of claim 29, wherein the nucleic acid encodes at least one other anticoagulant molecule.

Claim 44. (new) The method of claim 43, wherein the anticoagulant molecule is thrombomodulin or a functional fragment thereof.

Claim 45. (new) The method of claim 31, wherein the mammal is susceptible to an inflammatory or immunological stimulus and the method further comprises administering a therapeutic amount of at least one anti-coagulant, antithrombotic, or thrombolytic drug to treat or prevent that stimulus.

Claim 46. (new) The method of claim 45, wherein the drug is administered before step a) or after step c) of the method.

Claim 47. (new) The method of claim 46, wherein the anti-coagulant drug is coumadin.

Claim 48. (new) An engineered vascular graft produced by the method of claim 30.

Claim 49. (new) The engineered vascular graft of claim 48, wherein the vessel is an autologous saphenous vein graft (SVG).

Claim 50. (new) The engineered vascular graft of claim 48, wherein the vessel is an arterial graft.

Claim 51. (new) A kit for performing the methods of claims 29 or 30, the kit comprising:  
a) one or more of the agents for increasing the activated protein C (APC),  
b) means for detecting at least one of a) cell expression of the agents, and 2) the increased APC in the blood vessel; and  
c) directions for using the kit.